RESPONSE OF INTRACTABLE POST HERPETIC NEURALGIA TO INTRATECHAL BACLOFEN

Amr Hosny, MD, Thomas Simopoulos, MD, and Beth Collins, RN

An intractable case of Post-herpetic Neuralgia (PHN) in which all other treatment options were exhausted was successfully treated with intrathecal baclofen infusion with a complex continuous delivery mode.

A 72-year-old man presented to the pain clinic with a 4-year history of left lower extremity PHN. He had seen multiple experts in the field, failed numerous pharmacological therapies, and interventional techniques. After multiple neuraxial medications were tried, baclofen was chosen and an intrathecal drug delivery system was implanted. Eight months after the procedure he continues to have 80% pain relief.

Intrathecal baclofen (Lioresal Intrathecal, Medtronic Neurological, Minneapolis, MN) is a Food and Drug Administration (FDA)-approved therapy for medically intractable spasticity. The importance of gamma-aminobutyric acid (GABA) as an inhibitory neurotransmitter modulating either spasticity or persistent neuropathic pain states is without debate (1, 2). Animal data strongly supports the analgesic properties of intrathecal GABA agonists such as baclofen (3). Indeed there are a growing number of reports of intrathecal baclofen successfully used to manage chronic pain without spasticity (4, 5). In these reports, intrathecal baclofen did not produce intolerable side effects such as sedation or motor dysfunction (6). Here we present a case of refractory post-herpetic neuralgia (PHN) responding to intrathecal baclofen.

CASE REPORT

A 72-year-old male presented to our pain management center in 2001 with a 4-year history of left lower extremity pain secondary to PHN. This pain started 15 days after the development of acute herpes zoster in the context of a steroid treatment for Grave’s disease in 1997. His past medical history is significant only for one episode of atrial fibrillation. Despite initial acyclovir treatment, the pain had gradually worsened over the years. He described his pain as constant burning and throbbing with periods of sharp lancinations (as frequent as every 3 seconds) in the thigh that was exacerbated by walking or light contact with loose fitting pants. He reported improvement of pain when constant firm pressure was applied to his thigh. His pain would escalate with activity, and therefore significantly impacted his ambulation, thereby limiting his activities of daily living.

The physical examination revealed a healthy, well built but uncomfortable appearing elderly male. He was applying constant pressure to his left thigh. The sensory examination was without deficit to pin prick, and his reflexes were symmetrical (1/2) in the lower extremities. Motor examination was 5/5 in all major muscle groups in the lower extremities. He had marked hyperalgesia, hyperpathia and allodynia, most notable in the L3 distribution of the left lower extremity. The right lower extremity was unremarkable. There were healed zoster scars in the left L3 dermatome.

On presentation to the pain clinic he was on gabapentin 600 mg po tid, Synthroid, baclofen 20 mg po bid, low dose aspirin, multivitamins and zolpidem as a sleep aid. He reported some benefit from the gabapentin and baclofen at the current doses, but complained of side effects with increased dosage. His visual analog score (VAS) ranged from 7/10-10/10. Multiple narcotics, tricyclic antidepressants, anti-convulsants, topical agents, and anti-arrhythmic drugs were tried without benefit. He had also tried a TENS unit, selective nerve root blocks, lumbar sympathetic blocks, lumbar epidural steroids, and spinal cord stimulation, again without benefit.

Under our care, he received a series of 4 intrathecal steroid injections on a weekly basis with limited short-lived pain relief. After a lengthy discussion with the patient we decided to try neuraxial agents. An intrathecal catheter was inserted (L4/5) and he was admitted to the hospital for 3 days. Injection of intrathecal morphine (0.5 mg), hydromorphone (0.2 mg), as well as clonidine (30 mcg) on separate days, provided 30% pain relief, but they were associated with significant sedation and/or urinary retention. A trial of intrathecal baclofen as a 50 mcg bolus provided marked pain relief but again was associated with sedation. On the fifth day, just prior to removal of the catheter, a 25mcg bolus of baclofen was injected intrathecally with significant pain relief without sedation lasting 8 hours. Several weeks later, a 25 mcg subarachnoid injection of baclofen rendered similar effects on the pain level (VAS reduced by greater than 50%). The decision was made to implant an intrathecal drug delivery system.

A programmable pump (Medtron-
Spinal GABA release during SCS is one important neurochemical mechanism by which hyperexcitability is reduced in the dorsal horn of experimental animals (7). We hypothesized a lack of GABA due to inhibitory interneuron destruction during the initial zoster attack.

In fact, atrophy of the dorsal horn is known to occur in patients with long-term PHN (8). Many neurons in the spinal cord contain inhibitory neurotransmitters such as GABA (9). In animal studies, peripheral damage to primary afferent neurons results in transynaptic signs of degeneration of deafferentated spinal neurons in the dorsal horn (10). Cells that have undergone these changes are termed “dark neurons” (11). If loss of inhibitory interneurons occurs in PHN, then spontaneous activity of a disinhibited dorsal horn can cause pain. Support for this theory comes from animal nerve injury models, which are associated with coexisting spinal interneuron disruption, where it has been observed that baclofen reduces pain behaviors and allodynia (12). But it is unknown what types of neurons or neurotransmitters are lost in PHN patients. Suffice it to say that in many patients suffering from PHN, peripheral as well as central nervous system changes are thought to play a role in the persistence of pain.

Intrathecal baclofen has been reported effective in other refractory neuropathic pain states including complex regional pain syndrome, radicular pain, stump pain, and sciatic nerve injury (4, 6, 13). The authors of these cases observed a reduction in continuous and evoked pain (Chung model). They demonstrated that intrathecal baclofen reduces post nerve injury allodynia in rats without any apparent effect on motor function.

CONCLUSION

This case report lends more support to the observation that intrathecal baclofen is effective in some chronic neuropathic pain states that lack symptoms of spasticity. Our patient had failed multiple treatments and had suffered the torment of zoster related pain for many years. Intrathecal baclofen has been well established as safe and is FDA approved for intraspinal use but for spasticity only. Unlike morphine, which is FDA approved for intrathecal use for pain, baclofen is not associated with inflammatory catheter tip masses (17, 18). However abrupt intrathecal baclofen withdrawal can lead to life threatening sequelae and therefore the need for close long-term follow-up.

REFERENCES
